U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 29 April 2005

CASE NO. 2004-BLA-5603

In the Matter of

SHELBY J. SHEPPARD, Survivor of JOHN E. SHEPPARD, JR.,
Claimant

v.

BLUE SPRINGS COAL COMPANY,
Employer

and

DIRECTOR, OFFICE OF WORKERS COMPENSATION PROGRAMS,
Party-in-Interest

APPEARANCES:

Jason Huber, Esquire
Roger D. Forman, Esquire
Forman & Huber, LC
For the Claimant[1]

Mary Rich Maloy, Esquire
Jackson Kelly, PLLC
For the Employer

Before: RICHARD A. MORGAN
Administrative Law Judge

DECISION AND ORDER - DENYING BENEFITS

This proceeding arises from a claim for benefits filed by Shelby J. Sheppard, the surviving spouse of John E. Sheppard, Jr., a now deceased coal miner, under the Black Lung

[1] At the formal hearing, Jason Huber, Esquire, appeared on Claimant's behalf. Mr. Huber explained that he was substituting for Claimant's primary counsel, Roger D. Foreman, Esquire, who had a family medical emergency (TR 4). Most of the submissions, including the documentary evidence, motions, and Claimant's Closing Argument, were submitted by Roger D. Forman, Esquire.

Benefits Act, 30 U.S.C. §901, *et seq*. Regulations implementing the Act have been published by the Secretary of Labor in Title 20 of the Code of Federal Regulations.[2]

Black lung benefits are awarded to coal miners who are totally disabled by pneumoconiosis caused by inhalation of harmful dust in the course of coal mine employment and to the surviving dependents of coal miners whose death was caused by pneumoconiosis. Coal workers' pneumoconiosis is commonly known as black lung disease.

A formal hearing was held before the undersigned on October 28, 2004 in Charleston, West Virginia. At that time, all parties were afforded full opportunity to present evidence and argument as provided in the Act and the regulations issued. Pursuant to leave granted at the formal hearing, the record was initially held open until December 17, 2004 for the submission of closing arguments (TR 6).

By letter, dated December 8, 2004, Roger D. Foreman, Esquire (hereinafter "Claimant's counsel"), requested that Dr. Oesterling's deposition be received in evidence. In correspondence, dated December 15, 2004, Employer's counsel, objected to the admission of such evidence. On December 16, 2004, I issued an "Order Denying Claimant's Request for Post-Hearing Submission of Evidence." Subsequently, on December 22, 2004, a telephone conference call was held at which Claimant's counsel requested that I reconsider my ruling, and Employer's counsel maintained her objection to the admission of the post-hearing evidence. Thereafter, on December 23, 2004, I issued an "Order on Reconsideration Granting Claimant's Request for Post-Hearing Submission of Evidence." As stated therein, I marked and admitted Dr. Oesterling's deposition as Claimant's Exhibit 4 (EX 4). In addition, I provided the Employer until January 19, 2005 to submit a supplemental report by Dr. Oesterling as rehabilitative evidence. Pursuant thereto, I have marked and received Dr. Oesterling's supplemental report, dated January 14, 2004, as Employer's Exhibit 8 (EX 8). Moreover, I granted an extension to the respective parties, until January 28, 2005, for the submission of closing arguments.

In summary, the record includes the hearing transcript, Director's Exhibits 1 through 34 (DX 1-34), Claimant's Exhibits 1 through 4 (CX 1-4), and Employer's Exhibits 1, 2, 3, 4, 6, 7, and 8 (EX 1, 2, 3, 4, 6, 7, 8). On the other hand, Employer's Exhibit 5 (EX 5) has been excluded because it exceeds the evidentiary limitations set forth in the new regulations. Finally, I have also received and considered the pre-hearing reports and closing arguments of the respective parties.

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted and arguments made. Where pertinent, I have made credibility determinations concerning the evidence.

[2] The Secretary of Labor adopted amendments to the "Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969" as set forth in Federal Register/Vo. 65, No. 245 Wednesday, December 20, 2000. The revised Part 718 regulations became effective on January 19, 2001. Since the current claim was filed on October 17, 2002 (DX 7), the new regulations are applicable (DX 34).

Procedural History

On February 22, 1973, John E. Sheppard (a/k/a John E. Sheppard, Jr.), a former coal miner, filed an application for black lung benefits under the Act, which was repeatedly denied. Following the passage of the Black Lung Benefits Reform Act of 1977, Mr. Sheppard filed an election card on or about June 26, 1978, requesting review of his claim by the Social Security Administration. On April 18, 1979, the Social Security Administration denied the claim. Subsequently, the claim was also reviewed by the Department of Labor. On April 22, 1980, the Deputy Commissioner's office (now known as the District Director's office) also denied the claim. Since no appeal or further action was taken within one year of the April 22, 1980 denial, the above-referred miner's claim is finally closed (DX 1).

On May 17, 1984, the former miner filed a duplicate claim, which was denied by the District Director's office on January 17, 1985. Since no appeal or modification request was filed within one year of the May 17, 1984 denial, the above-referred miner's claim is also finally closed (DX 2).

On January 18, 1986, Mr. Sheppard filed a third claim. Following a formal hearing, held on June 17, 1990, Administrative Law Judge David A. Clarke, Jr., issued a Decision and Order-Rejection of Claim, dated August 2, 1990. Since no appeal or other action was taken within one year of Judge Clarke's decision, the 1986 claim is also finally denied and administratively closed (DX 3).

On July 28, 1997, the former miner filed a fourth application for benefits under the Act, which was denied by the District Director's office on December 29, 1997. By letter, dated March 17, 1998, the District Director confirmed that Claimant had not pursued the claim. Since no appeal or modification request was filed within one year of the foregoing denial, the miner's fourth claim is also finally denied and administratively closed (DX 4).

On January 25, 2000, Mr. Sheppard filed his fifth (and final) claim under the Act. Following a formal hearing on August 27, 2001, I issued a Decision and Order Denying Benefits, dated November 20, 2001. Following the miner's timely appeal, and while the claim was still pending before the Benefits Review Board, Mr. Sheppard passed away. Accordingly, on October 18, 2002, the Claimant, Shelby J. Sheppard, requested that she be considered as a substitute party on behalf of her husband concerning his pending claim before the Benefits Review Board (DX 5).

A "Petition for Modification" was filed on behalf of John E. Sheppard by Roger D. Forman, Esquire (DX 5). The Petition for Modification, which was filed on or about November 27, 2002, requested that the case be remanded from the Benefits Review Board for modification based upon an autopsy finding of complicated pneumoconiosis and the miner's death certificate. However, the Benefits Review Board declined to remand the case. To the contrary, the Board issued a Decision and Order, dated December 16, 2002, in which it affirmed my Decision and Order denying benefits, while also noting that Mr. Sheppard had died on September 5, 2002, and that his widow had notified the Board on October 18, 2002 that she would be pursuing his pending claim. (DX 5, BRB Decision and Order, dated 12/16/02, note 1).

Under cover letter, dated December 26, 2002, Mr. Forman filed a document entitled "Petitioner's Motion for Reconsideration of Decision Denying Benefits Dated December 16, 2002," on behalf of the deceased miner, which expressly cited the Modification Petition (DX 5). Subsequently, on March 19, 2003, the Benefits Review Board issued an Order of Reconsideration, in which the motion for reconsideration was denied. Furthermore, the Board stated: "Further review of the disposition of the instant case may be obtained by appeal to the United States Court of Appeals of the appropriate circuit." (DX 5, BRB Order on Reconsideration, dated 3/19/03, note 1). Since no further appeal or modification request was filed within one year of the Board's Order on Reconsideration, dated March 19, 2003, the miner's fifth claim is also finally denied and administratively closed (DX 5). Accordingly, the only matter under consideration herein is the survivor's claim.[3]

On September 5, 2002, John E. Sheppard, Jr., passed away (DX 15). On October 17, 2002, Claimant, Shelby J. Sheppard, filed the current application for black lung benefits under the Act, as his surviving spouse (DX 7). This claim was granted by the District Director in his Proposed Decision and Order, dated September 5, 2003 (DX 26). Following Employer's timely request for a formal hearing (DX 27), this matter was referred to the Office of Administrative Law Judges on or about January 23, 2004 for *de novo* adjudication. As stated above, the formal hearing was held on October 28, 2004, and the record was held open until January 28, 2005.

Issues

The primary issue is whether the miner's death was due to pneumoconiosis, as defined in the Act and applicable regulations (*See* Employer's Closing Argument).[4]

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background and Employment History

A. Coal Miner and Length of Coal Mine Employment

Claimant's testimony at the formal hearing was vague regarding the extent her husband's coal mine employment history (TR 18-20). However, I find that the documentary evidence and Mr. Sheppard's testimony at prior hearings establish that he engaged in coal mine employment for at least 33 years ending in 1984 (DX 1-5, 8-11). Similarly, the District Director found that Claimant has proven 33 years of coal mine employment (DX 26).

B Date of Filing

[3] Although not controlling, the District Director included a Memorandum to File which also states that all five of the miner's claims are administratively closed and not subject to adjudication. Furthermore, as discussed above, in conjunction with the survivor's claim, I accord little weight to the medical evidence submitted in support of the miner's Petition for Modification.

[4] At the formal hearing, Employer cited various other contested issues (TR 6-7). However, as discussed above, the pathology and recent medical opinion evidence clearly establishes the presence of pneumoconiosis. Furthermore, Mr. Sheppard engaged in coal mine employment for considerably more than ten years, and Employer failed to rebut the presumption that that the disease arose from such employment. *See* 20 C.F.R. §§ 718.203 and 718.302.

Claimant, Shelby J. Sheppard, filed her claim for survivor's benefits under the Act, on October 17, 2002 (DX 7). Employer has stipulated, and I find, that the claim was timely filed (DX 32).

C. Responsible Operator

Although the evidence is conflicting regarding the exact period of time in which Mr. Sheppard's was employed by Blue Springs Coal Company (*Compare* DX 8, 10), the record clearly establishes that he worked for Employer for more than one calendar year. Moreover, the case file also establishes that Mr. Sheppard's last coal mine employment was with Employer (DX 1-5, 8-11). Accordingly, I find that Blue Springs Coal Company is the properly designated responsible operator.

D. Dependents

Claimant, Shelby J. Sheppard, has one dependent for purposes of augmentation of benefits under the Act; namely, her younger daughter, Phyllis M. Sheppard, who was born on August 10, 1984, and is a college student (DX 7, 13, 14; TR 17-18).

E. Personal Background and History

The former miner, John E. Sheppard, Jr., was born on May 28, 2001. He married Shelby J. Sheppard (nee Collins) on September 25, 1954. They remained married until the miner's death on September 5, 2002. Claimant has not re-married since her husband's death (DX 7, 12, 15; TR 17).

Claimant testified that her husband was already a cigarette smoker when they met in 1954, but that she didn't know when he first began smoking. Claimant acknowledged that he continued smoking until 2001 (TR 20-21). Furthermore, the medical evidence, including the reports previously submitted in conjunction with the miner's prior claims, establish that Mr. Sheppard began smoking cigarettes when he was a boy, and that he continued for almost his entire life (DX 1-5).

Claimant's daughter, Terena Surgoine, also testified at the formal hearing (TR 12-13). Although she is a Registered Nurse, Ms. Surgoine stated that she was testifying in a *non*-medical capacity (TR 13). However, Ms. Surgoine's testimony primarily relates to conversations with Dr. Figueroa, who performed surgery on her father on August 9, 2002 (TR 13-16; *see also* CX 2). Ms. Surgoine stated that Dr. Figueroa performed a "right Thorocodomy (sic) and a lower lobe lumbexomy (sic)" on August 9, 2002, and that he spoke to the family, including herself, in the waiting room following the surgery. Over Employer counsel's objections, I allowed the following exchange between Claimant's counsel and Ms. Surgoine:

Q Tell me what he [Dr. Figueroa] said about the surgical procedure.

A When he came out from the surgery, he said that dad was doing well, that they did remove the lower lobe, and that the pathologist at the time said that the margins were clear around…

Q What does that mean when he said the margins are clear?

A I think they allow just a certain amount of tissue surrounding the tumor, and if those margins are clear, they consider it as getting all of the tumor.

Q Is that your impression from the conversation with the doctor?

A Yes.

Q That they removed the entire tumor?

A Right – that they removed the lower lobe of the right lung and that there was, to his knowledge, what the pathologist's report would be was (sic) at that time, they felt like they had gotten it all, but they would send it off to Pathology to get a further report.

Q What if anything, did he say to you about the state of your father's lungs?

A He said that the procedure took a little longer than normal because, he said, dad's lungs – when he got in there and started to make an incision and do the dissection – were hard. It was like cutting through rock – that he clearly saw coal dust deposits within his lung, and even when he went to remove them, the lymph nodes – some of those were black and hard to remove. It just took a little longer because of the condition of dad's lung.

Q It is my understanding that at some point, your father had some tubes inserted in his lungs?

A After a Thorocodomy (sic), they will insert chest tubes. He had two Plural (sic) chest tubes, and he was in the hospital several weeks. He did not come until the 24th, and he came home with those chest tubes. I was real concerned because – usually being a nurse and seeing a Thorocodomy (sic) – usually these chest tubes are out within three to five days, and Dad's continued, and he was having problems with infection and so forth. It was his opinion…

Q The doctor?

A Yes, the doctor, Dr. Figuroia (sic) – that his lung was not sealing well because of the scarring of his lungs from the coal dust. This is the statement he made to be (sic), because I was concerned about him and why he could not get rid of the chest tubes. And

he said that he felt like that was part of the problem – that his lungs were not sealing because of the scarring.

Q When did he say that?

A This was a couple of weeks after the surgery that he told me that.

Q When did your father die?

A He died on September the 5th 2002, and he still had the chest tubes.

(TR 14-16).

Medical Evidence

Except as modified or superseded herein, all of the medical evidence summarized in my Decision and Order Denying Benefits, dated November 20, 2001, as affirmed by the Benefits Review Board, in its Decision and Order, dated December 16, 2002, is incorporated by reference herein. Based upon the evidence previously presented, I found that Mr. Sheppard suffered from a total respiratory disability. However, I denied benefits based upon the miner's failure to establish the presence of pneumoconiosis and/or that his total respiratory disability was due to pneumoconiosis. On appeal, the Board affirmed my findings that pneumoconiosis had not been established under §718.202(a)(1), (2), and/or (3). (DX 5, BRB Decision and Order, pp. 2-4, *see also* note 3). However, the Board found it unnecessary to consider Claimant's argument regarding the "pneumoconiosis" issue under §718.202(a)(4), because it affirmed the denial of benefits based upon the miner's failure to establish that pneumoconiosis substantially contributed to his disability pursuant to §718.204(c). (DX 5, BRB Decision and Order, pp. 4-5).

As discussed herein, this is a survivor's claim. Accordingly, "total disability" and "disability causation" are not necessary elements of entitlement herein. Furthermore, as previously noted, and as summarized below, the autopsy evidence and more recent medical opinion evidence clearly establishes the presence of pneumoconiosis. Moreover, the presumption contained in §718.203 and §718.302, which establishes the relationship between the miner's pneumoconiosis and his more than ten years of coal mine employment, has not been rebutted. Therefore, the crux of this case is whether Claimant has established "death due to pneumoconiosis" pursuant to §718.205(c), which also includes the sub-issue of whether Claimant has established the presence of *complicated* pneumoconiosis.

In addition to the extensive medical data contained in the multiple (closed) miner's claims, the record includes the following evidence, which was submitted in conjunction with the survivor's claim: the miner's death certificate (DX 15) and the recent medical opinions of Drs. Jelic (DX 17), Figueroa (CX 2), Oesterling (EX 1, 8; CX 4), Bush (EX 2), Jarboe (EX 4), Rasmussen (CX 1), and Zaldivar (EX 3, 6, 7), respectively.

The miner's death certificate, which was signed by Dr. E. Figueroa, states that Mr. Sheppard died on September 5, 2002, at age 71 (DX 15). The immediate cause of death was

reported as "respiratory failure" due to "Adult Respiratory Distress Syndrome" due to "Bronchopneumonia." In addition, the following other significant conditions were listed, which reportedly contributed to death, albeit not to the underlying cause cited above: "History of lung cancer & pneumoconiosis." Although the death certificate states that an autopsy of the lungs was performed, it is unclear whether the autopsy findings were available prior to the completion of the death certificate (DX 15).

The record contains an "AMENDED" Autopsy Pathology Report, dated October 22, 2002, by Dr. Tomislav M. Jelic (DX 17). It indicates that the original report was amended on October 22, 2002 by Donna J. Doles, in order to make a "correction in gross description" (DX 17, p. 2). It also lists Dr. Edmundo E. Figueroa as the surgeon (DX 17, p. 1). Dr. Jelic reported the following "Gross Description" of the lungs:

> LUNGS: The right lung weighs 840 grams and the left lung weighs 1180 grams. The right lower lobe is surgically absent. The serosal surface is gray and shows anthracotic pattern. There are no subpleural emphysematous bullae. The lungs are subcrepitant throughout and on cut section the parenchyma and the left lower lobe exhibits bright red discoloration.. Black dots are present throughout the both lungs, especially in both upper lobes.

(DX 17, p. 2). In addition, Dr. Jelic set forth the following "Microscopic Description:"

> Sections of the lungs demonstrate coal workers' pneumoconiosis with focal features of progressive massive fibrosis, thus, complicated coal workers' pneumoconiosis is present. Numerous macules and frequent anthracosilicotic nodules are noted in all lung lobes. Peribronchial lymph nodes are replaced with merging anthracosilicotic nodules. In the macules and in the anthracosilicotic nodules of the lung parenchyma and lymph nodes, numerous polarized particles consistent with silica particles are present.
>
> Acute and organizing bronchopneumonia are present in all lung lobes. The right middle lobe exhibits acute fibrinopurulent pleuritis. Acute bronchopneumonia is characterized with abundance of neutrophils in the alveoli, and organizing bronchopneumonia is featured with residual inflammatory cells intermixed with young fibroblasts, collagen fibrils, histiocytes, pneumocytes type 2, and hemosiderin laden macrophages. Gram stain demonstrates gram positive cocci. GMS stain for fungi is negative.
>
> Organizing pulmonary emboli are present in the large and medium size pulmonary arteries in the left lower lobe and right middle lobe.
>
> Emphysema is marked. Chronic bronchitis presents with reversed serous to mucinous bronchial gland ratio (1:2), partial mucinous metaplasia of the respiratory epithelium, and infiltration of the bronchial glands with small lymphocytes and occasional lymphoid aggregates. Acute exacerbation of the chronic bronchitis is features (sic) with abundance of neutrophils in the mucus lining bronchial respiratory epithelium. Iron stain has not demonstrated asbestos bodies.

(DX 17, pp. 2-3). Furthermore, Dr. Jelic set forth the following clinical pathologic summary:

> Mr. Sheppard John E. was a 71 year old retired coal miner who had protracted pneumonia not responding to antibiotic treatment. Further work up including CT of the chest demonstrated mass in the right lower lobe. Lobectomy showed moderately differentiated squamous cell carcinoma, coal workers' pneumoconiosis, and chronic silicosis (SP02-18552). Despite intensive postoperative treatment the patient expired and his Family requested autopsy limited to the lungs.
>
> His previous medical history included myocardial infarction in 1980, cerebrovascular accident with no sequela, remote peptic ulcer, gastroesophageal reflux, chronic obstructive pulmonary disease, coal workers' pneumoconiosis, and benign prostate hypertrophy. He worked as a coal miner for 34 years.
>
> An autopsy documents complicated coal workers' pneumoconiosis also known as progressive massive pulmonary fibrosis, chronic silicosis, gram positive cocci acute, and organizing bronchopneumonia involving all residual lung lobes, acute fibrinopurulent pleuritis in the right upper and middle lobes as well as organizing pulmonary emboli in the large and medium size pulmonary arteries of the left lower and right middle lobe. Emphysema is marked and chronic bronchitis exhibits acute exacerbation.
>
> The patient died because of lung cancer complicated with pulmonary embolizations, extensive acute and organizing bronchopneumonia, and acute fibrinopurulent pleuritis. Complicated coal workers' pneumoconiosis, marked emphysema and acute exacerbation of chronic bronchitis were contributory factors.

(DX 17, pp. 1-2). Based upon the foregoing, Dr. Jelic set forth the following diagnoses:

I. Status post recent right lower lung lobe lobectomy because of moderately differentiated squamous cell carcinoma, TNM IB, T2, N0, MX (SP02-18552).

II. No residual cancer seen.

III. Gram positive cocci acute, and organizing bronchopneumonia involving all lung lobes.
   A. Acute fibrinopurulent pleuritis involving right upper and middle lobes.

IV. Pulmonary emboli.

V. Complicated coal workers' pneumoconiosis (massive pulmonary fibrosis).
   A. Chronic silicosis.

V. Marked emphysema.

VI. Acute exacerbation of chronic bronchitis.

(DX 17, p. 1).

Dr. Edmundo E. Figueroa issued a cursory, handwritten report, dated October 7, 2004 (CX 2). As stated above, Dr. Figueroa is the physician who signed the miner's death certificate (DX 15). Furthermore, he performed the thoracotomy and, apparently, was also the surgeon for the miner's autopsy (CX 2; DX 17, p. 1). The full text of Dr. Figueroa's report is as follows:

> I did the right thoracotomy on the late John E. Sheppard on Aug. 9, 2002. At that time I noticed gross and advanced changes of coal worker's pneumoconiosis in the whole right lung.

(CX 2).

Dr. Everett F. Oesterling, who is Board-certified in Anatomical Pathology, Clinical Pathology, and Nuclear Medicine, issued a report, dated April 20, 2004, in which he reviewed medical data supplied by Employer's representative, and analyzed the histologic slides (EX 1). In order to illustrate and document his findings, Dr. Oesterling utilized photomicrophages, which he referred to in his detailed report. Following Dr. Oesterling's analysis of the pathology evidence (EX 1, pp. 1-4),[5] Dr. Oesterling stated:

> At this point I would like to address, at least in part, the questions which you (Employer's representative) posed and would state the following with reasonable medical certainty:
>
> 1. This gentleman's lung tissue does show evidence of coalworkers' (sic) pneumoconiosis in the form of a relatively mild micronodular coal workers' (sic) pneumoconiosis.
>
> 2. The limited changes due to dust exposure appear insufficient to have in any way contributed to this gentleman's death.
>
> 3. This gentleman did experience significant respiratory impairment prior to his death, but not due to mine dust exposure.
>
> 4. In all probability due to the total combination of disease processes in his lungs, he was a respiratory cripple and therefore would have been disabled prior to this (sic) death, but not on the basis of mine dust exposure.
>
> 5. Coal mine dust exposure did not contribute to pulmonary or respiratory impairment or any form of disability in this claimant.
>
> 6. Again coalworkers' (sic) pneumoconiosis and mine dust exposure was in no way a factor in hastening this gentleman's death.

[5] The page numbers cited herein refer to those in Dr. Oesterling's report, and not the numbers listed on Employer's Exhibit 1. Employer's counsel included her cover letter, dated April 28, 2004, as page 1 of Employer's Exhibit 1, thereby confusing the issue.

(EX 1, pp. 4-5). Following his further discussion of the histologic slides, in conjunction with other clinical data, the miner's smoking history commencing at age 12 and continuing throughout much of his adult life, the removal of 20% of the miner's lung for smoking induced squamous cell carcinoma, citation and inclusion of medical literature of Dr. Spencer regarding the pathology of the lung, and Claimant's past history of myocardial infarct (EX 1, pp. 5-7), Dr. Oesterling stated:

> In concluding I would restate with reasonable medical certainty that his (sic) unfortunate gentleman had minimal micronodular coalworkers' (sic) pneumoconiosis, a level of disease which appears insufficient to have in any way contributed to lifetime symptomatology, lifetime disability, and in no way did it contribute to, hasten, or cause his death.

(EX 1, p. 7).

Dr. Oesterling also testified at a deposition held on October 20, 2004 (CX 4). Dr. Oesterling stated that he has extensive general experience in anatomical pathology, clinical pathology, and nuclear medicine, and, in particular, expertise in lung pathology (CX 4, pp. 3-12). However, Dr. Oesterling also acknowledged that a significant part of his current income involves reviewing cases for coal mine operators. Although he initially started doing cases for representatives of the United Mine Workers, 100% of the cases he has received for the last 15 to 20 years are from defendants. In addition, Dr. Oesterling stated that his total fee in this case is $2,500 (CX 4, pp. 44-46). In his substantive testimony, Dr. Oesterling discussed the evidence, in particular his pathology findings (CX 4, pp. 12-44). Based upon the foregoing, Dr. Oesterling reiterated that the amount of dust shown on autopsy was not significant; that the changes seen on autopsy are not those of progressive massive fibrosis; and, that neither mine dust nor coal worker's pneumoconiosis contributed or hasten the miner's death (CX 4, pp. 42-44). I note, however, that Dr. Oesterling's deposition testimony also included some statements which were somewhat ambiguous. For example, Dr. Oesterling acknowledged that he found one lesion which was about 1.4 cm, and that, "sizewise" it would meet the classification for complicated coal worker's pneumoconiosis. In addition, he conceded that there was one other lymph node which "approached the centimeter," which would have been conceivably described as progressive massive fibrosis (CX 4, p. 50). Furthermore, Dr. Oesterling acknowledged that there is some silica which has produced areas of fibrosis (CX 4, p. 54). However, Dr. Oesterling also testified that the larger nodules are found in the hilar lymph node, not in the lung. Furthermore, Dr. Oesterling stated that a reaction to coal dust in the lymph node, by definition, is not coal worker's pneumoconiosis (CX 4, pp. 20-22). In addition, Dr. Oesterling explained that the predominant pigment in the mass, found in the lymph nodes, is hemosiderin which does not entrap mine dust (CX 4, p. 22). Moreover, Dr. Oesterling further explained that the central core of the mass is hemosiderin, and that black pigment is on the periphery and is not part of the fibrotic process that comprises the central core of the nodule (CX 4, pp. 54-56). Dr. Oesterling also acknowledged that many years ago the miner's bronchitis was probably related to his coal mine dust exposure; that coal mine dust can be a contributing factor to bronchitis, particularly during periods of active mining, and that bronchitis would have contributed to an unspecified extent to the process which culminated in the miner's death (CX 4, pp. 70, 73-74). On the other hand, Dr. Oesterling also testified that Mr. Sheppard left coal mine employment many years ago

but continued to smoke. Accordingly, Dr. Oesterling attributed the miner's bronchitis to his extensive smoking history (CX 4, pp. 71-72). In summary, Dr. Oesterling stated that, although coal mine dust at some point contributed to the miner's chronic bronchitis, its effect stopped following his removal from the mines, and that the miner's death was due to other factors, not mine dust exposure (CX 4, pp. 74-75).

In a supplemental report, dated January 14, 2005 (EX 8), Dr. Oesterling sought to clarify his opinion, as previously set forth in his prior report and deposition testimony. Citing the deposition transcript, Dr. Oesterling provided the following explanation of his testimony:

1. In my testimony on pages 47-60 I have attempted to describe processes which might be misconstrued as complicated coalworkers' (sic) pneumoconiosis or progressive massive fibrosis, however I believe that I have also very carefully tried to illustrate and explain that coal mine dust was not a major contributing cause of the fibrotic processes which are present. These lesions have resulted from extensive deposition of another fibrogenic substance, hemosiderin, a breakdown byproduct of hemoglobin which has been released from red blood cells that have been extravasated into the air spaces. This process is related to this gentleman's passive congestion which resulted from his extensive cardiac disease. Mine dust has been entrapped in the process, but it was not the initiating or causative agent.

2. On pages 70-75 I was questioned and attempted to answer the issue of chronic bronchitis. It is my firm belief that the primary cause of this gentleman's chronic bronchitis was his early onset of smoking and his continued smoking habit following the termination of his employment in the mines. By contrast, any limited component that mine dust contributed to the evolution of his bronchitis would have terminated within a relatively brief period following his cessation of exposure to coalmine dust. Conversely he continued to inhale cigarette smoke and therefore the continued bronchitis can be related purely to his habit and not his prior employment. His chronic bronchitis was not a contributing or hastening factor in his death since the primary cause was related to his cardiac status and the extensive destruction of his lungs due to the marked passive congestion and hemosiderosis superimposed upon his preexistent panlobular emphysema, a condition unrelated to mine dust exposure.

3. Coal dust exposure and coalworkers' (sic) pneumoconiosis in no way played any role nor did it in any way contribute to this gentleman's primary pulmonary impairment or his demise.

(EX 8).

Dr. Stephen T. Bush, who is Board-certified in Anatomic and Clinical Pathology and in Medical Microbiology, issued a report, dated July 26, 2004 (EX 2), in which he discussed various medical data and other documents provided by Employer's representative. Dr. Bush stated that after working as underground coal miner for 23 years, Mr. Sheppard worked an additional 10 years as a foreman on light duty because of shortness of breath until 1984, when he was laid off. Dr. Bush also reported that Claimant was a life-long cigarette smoker, alleging ½

pack per day until late in life when this rate diminished. Following his discussion of various pulmonary evaluations and other medical records, and his analysis of the histologic slides, Dr. Bush stated:

1. The lungs show evidence of a mild degree of simple coal worker's pneumoconiosis. The autopsy prosector noted grossly that the cut surface of the lungs appeared bright red with "black dots." This description corresponds to the histologic findings of a very small amount of black dust pigment consistent with coal dust associated with a fibrous reaction forming coal worker micronodules within the parenchyma measuring up to 0.4 cm. These lesions are surrounded by a limited amount of focal dust emphysema.

   Exceptional larger nodules in hilar lymph nodes measuring up to 1.2 x 0.6 cm contain prominent amounts of birefringent particles of silica and silicates associated with a very small amount of black dust pigment. The largest of these nodules is noted in the subpleural area. These correspond to anthrasilicotic nodules, some of which have central degeneration, one of which has localized ossification indicating the ancient and indolent nature of these lesions. There is no evidence of a proliferative fibrous reaction but, a bland accellular composition. No more than 5 percent of the lung sections are involved by coal worker nodules or anthrasilicotic lesions, none of which have the massive fibrous proliferation with dense pigmentation characterizing progressive massive fibrosis.

   The histologic slides show centrilobular emphysema not related to fibrosis or coal dust pigment deposition. The emphysema is typical of that noted in heavy cigarette smokers.

   Although no parenchyma is represented on the histologic slides. (sic) Changes of adult respiratory distress syndrome in proliferative phase explains the intra-alveolar fibrohistiocytes. Some slides show accumulations of fibrin on the pleural surface. Several slides show thrombi in medium and large size vessels and one slide shows pulmonary infarction. Localized acute bronchopneumonia is evident on a few slides.

2. The coal worker's pneumoconiosis did not contribute to death. Death apparently resulted from severe pulmonary disease secondary to lung damage attributable to a high degree of certainty to the effects of smoking: carcinoma of the lung necessitating right upper lobe resection decreasing the availability of lung tissue for vital functioning, severe centrilobular emphysema and chronic bronchitis. These changes are complicated by pulmonary emboli and infarction, localized acute bronchopneumonia and changes of adult respiratory distress syndrome.

3. Mr. Sheppard suffered from respiratory impairment as a result of severe centrilobular emphysema and carcinoma of the lung necessitating lung resection. The small amount of lung tissue affected by the changes of coal worker's

pneumoconiosis is too limited in degree and extent to have caused or contributed to respiratory impairment.

4. Mr. Sheppard was totally disabled prior to death as a result of the pulmonary diseases resulting from cigarette smoking and from cardiac disease clinically diagnosed.

5. Coal worker's pneumoconiosis or occupational exposure to coal dust did not contribute in any way to respiratory impairment or disability suffered by Mr. Sheppard. The coal dust disease was so limited in degree and extent that it would not have produced signs or symptoms of respiratory disease by itself, nor could it have contributed in any significant way to the fatal illness of Mr. Sheppard.

6. Coal worker's pneumoconiosis or coal mine dust exposure played no role in nor hastened the death of Mr. Sheppard. Mr. Sheppard would have died at the same time and in the same manner if he had never been exposed to the pulmonary hazards of coal mine employment.

(EX 2, pp. 4-5).

Dr. Bush also discussed the opinions of the autopsy prosector, as well as those of Drs. Oesterling and Rasmussen, respectively. In summary, Dr. Bush disagreed with the autopsy prosector's finding of complicated coal worker's pneumoconiosis, noting that the largest lesions attributable to coal mine employment are anthrasilicotic nodules in the lymph nodes with only one in the lung substance located beneath the pleura. Moreover, Dr. Bush reiterated that these lesions are indolent and acellular with no evidence of a proliferating component to indicate a progressive process. In most other aspects, Dr. Bush agreed with the prosector's findings. Furthermore, Dr. Bush essentially agreed with Dr. Oesterling, but noted that he erroneously stated that the left lower lobe had been removed rather than the right lower lobe. Dr. Bush also agreed with parts of Dr. Rasmussen's opinion; namely, that coal worker's pneumoconiosis may progress after cessation of coal mine employment and emphysema of some degree may be associated with coal worker's pneumoconiosis. However, Dr. Bush reiterated: "Mr. Sheppard had a mild degree of simple coal worker's pneumoconiosis and a severe degree of centrilobular emphysema resulting entirely from a lifelong habit of cigarette smoking. I disagree with the conclusion of Dr. Rasmussen that coal mine dust exposure played a role in pulmonary impairment, disability, and death in Mr. Sheppard." (EX 2, pp. 5-6).

Dr. Thomas M. Jarboe, a B-reader who is Board-certified in Internal Medicine and Pulmonary Disease, had provided reports in conjunction with the miner's case.[6] Dr. Jarboe also issued a report, dated August 9, 2004, regarding the survivor's claim, in which he reviewed additional records provided by Employer's representative (EX 4). Following his discussion and analysis of such evidence, Dr. Jarboe responded to various questions posed by Employer's

[6] Based upon the, then, available medical evidence, Dr. Jarboe found that Mr. Sheppard suffered from a totally disabling respiratory impairment. However, Dr. Jarboe did not find sufficient evidence to justify a diagnosis of pneumoconiosis. Furthermore, he found that the miner's total disability was not related to coal dust inhalation (DX 5).

counsel. In summary, Dr. Jarboe stated: 1. Based upon the medical records, and, in particular, Dr. Oesterling's descriptions of the lung tissues, Mr. Sheppard had simple micronodular coal worker's pneumoconiosis, *not* complicated pneumoconiosis. 2. Mr. Sheppard had a severe pulmonary impairment attributable to factors other than simple coal worker's pneumoconiosis, namely, smoking-induced panlobular emphysema, hemosiderosis, the removal of the miner's right lung due to carcinoma, and pulmonary emboli, which are all unrelated to pneumoconiosis. 3. From a respiratory or pulmonary standpoint, the miner suffered from a reduced capacity to perform his coal mine employment prior to his death. 4. As a whole man, the miner suffered from a reduced capacity to perform his coal mine employment prior to his death. 5. Mr. Sheppard's coal dust exposure did not play any role in the miner's disability prior to his death. 6. Neither coal worker's pneumoconiosis nor coal dust exposure played any role in or hastened the miner's death. 7. His opinion would not change regarding the absence of any role of coal dust exposure in Mr. Sheppard's disability or death, even if the miner were found to have pneumoconiosis, and, in fact, Mr. Sheppard does have simple coal worker's pneumoconiosis (EX 4, pp. 5-9).

Dr. Donald L. Rasmussen, a B-reader who is Board-certified in Internal Medicine, Forensic Examiners, and Forensic Medicine, as well as a Senior Disability Analyst with extensive experience in pulmonary medicine, had provided various reports in conjunction with the miner's claims (DX 5).[7] Dr. Rasmussen also issued a report, dated October 7, 2004, in conjunction with the survivor's claim, in which he reviewed various medical data and also cited medical literature (CX 1). Dr. Rasmussen cited the opinions of Drs. Zaldivar and Jarboe who found that Mr. Sheppard's death was wholly unrelated to coal mine dust exposure, as well as the opinions of Drs. Oesterling and Bush whom he said reached the same conclusion, based upon their finding that the pneumoconiosis was not extensive. Moreover, Dr. Rasmussen described Drs. Oesterling and Bush as "highly competent pathologists," and Dr. Rasmussen acknowledged "I am not a pathologist and I cannot discuss the anatomical changes with any degree of competence." Notwithstanding the foregoing, Dr. Rasmussen stated: "the conclusion that disability and death can occur only in the presence of widespread and extensive pneumoconiosis is not born (sic) out by the multiple epidemiologic and mortality studies which have been performed in multiple countries throughout the world." Subsequently, Dr. Rasmussen cited various articles which discuss the following issues: honeycombing of the lungs, as shown on the surgical pathology report, as an indicator of severe lung disease and also noted in coal workers; unknown morphologic relationships concerning emphysema and chronic obstructive lung disease, whereby coal miners may lose lung function and develop disabling chronic obstructive lung even with negative x-ray evidence of pneumoconiosis, and which relates centracinar and panacinar emphysema to coal mine dust exposure; epidemiologic studies indicating patients do not have to suffer from complicated coal worker's pneumoconiosis to develop disabling or fatal lung disease and that miners may become disabled secondary to dust exposure independent of x-ray changes; and, mortality studies indicating that coal miners die excessively of chronic lung disease independent of x-ray changes. In summary, Dr. Rasmussen stated:

[7] In his most recent report in the living miner's case, dated July 27, 2001, Dr. Rasmussen reviewed the, then, available evidence and opined that the miner suffered from coal worker's pneumoconiosis, as well as chronic disabling disease of the lung. Furthermore, Dr. Rasmussen related the latter condition to coal mine dust exposure and cigarette smoking (DX 5).

Based on all of the above, there is no truly scientific basis for concluding that coal mine dust played no role in Mr. Sheppard's disabling and ultimately fatal chronic lung disease. In this connection, it is well known that many coal miners develop very pronounced x-ray abnormalities including complicated pneumoconiosis with no measurable physiologic impairment.

In addition to the epidemiologic studies, I can also review my own 40+ years of evaluating coal miners to note that it is by no means uncommon for coal miners to have severe impairment with even physiologic parameters with or without airway obstruction, who have minimal or in fact no x-ray changes consistent with pneumoconiosis.

Based on all of the above, it is my opinion to a reasonable degree of medical certainty that Mr. John Sheppard suffered from a chronic dust disease of the lung including coalworkers' (sic) pneumoconiosis and silicosis, which arose from his coal mine employment and which was a significant contributing factor to his disabling lung disease and ultimately to his death.

(CX 1, p. 4).

Dr. George L. Zaldivar is a B-reader, who is Board-certified in Pulmonary Disease, Internal Medicine, Sleep Disorder, and Critical Care Medicine (EX 3). Dr. Zaldivar had provided his medical opinion in the miner's case (DX 5).[8] Dr. Zaldivar also issued a report, dated July 28, 2004, in conjunction with the survivor's claim, in which he reviewed various additional medical data and cited medical literature (EX 3). Following his discussion and analysis, Dr. Zaldivar stated:

OPINION

Taking all this information into consideration, my answers to your [Employer's representative] questions are as follows:

1. There is sufficient objective evidence to justify a diagnosis of coal workers' pneumoconiosis in this man.

2. There was a respiratory impairment present prior to his death. This pulmonary impairment was the result of emphysema caused by his lifelong history of smoking and immediately prior to death finding of pulmonary emboli, which further compromised his oxygenation. Additionally, there was the burden of resection of a portion of his lung, which further compromised his ventilation and ability to oxygenate. All of this resulted in severe respiratory compromise.

[8] Based upon the, then, available medical evidence, Dr. Zaldivar made an x-ray finding of sarcoidosis, which was not credited. In addition, Dr. Zaldivar found that Mr. Sheppard suffered from a totally disabling respiratory impairment. In conclusion, Dr. Zaldivar opined that there was no medical evidence to justify a diagnosis of pneumoconiosis or any dust-related disease of the lungs, and that the miner's total disability was not related to pneumoconiosis or coal dust inhalation (DX 5).

3. From the pulmonary standpoint, prior to his death and even at the time of my examination of Mr. Sheppard in the year 2000, he was incapable of performing his usual coal mining work or work requiring similar activity and such impairment was the result of emphysema.

4. Coal dust exposure did not play any role in his disability prior to his death.

5. The coal workers' pneumoconiosis found by the pathologist did not play any role in his death nor did it hasten his death for the reasons I have mentioned.

6. According to all of these documents, which included examinations, pathology reports, etc, Mr. Sheppard's death would have occurred when and as it did even if he had never worked in the coal mines. The pneumoconiosis found by the pathologists was, as described by Dr. Oesterling, an incidental finding at autopsy. They represented a marker of Mr. Sheppard's occupation but did not cause any lung pathology that could have resulted in a physiological impairment.

(EX 3, pp. 10-11).

Dr. Zaldivar also testified at a deposition held on October 5, 2004 (EX 6). Dr. Zaldivar stated that he has approximately 30 years of experience as a B-reader and Board-certified pulmonary specialist, and continues to evaluate coal miners and other individuals who may have occupational lung disease. The main part of his medical practice entails taking care of patients and evaluating patients at their request or at the request of their physicians regarding pulmonary problems or conditions, both in his office and in the hospital. Accordingly, he has seen a variety of pulmonary conditions. In addition, part of his practice involves evaluating and treating sleep disorders. Dr. Zaldivar acknowledged that, in terms of litigation, he has been hired by coal mine operators about 95% of the time, and the remaining 5% he has been employed by the Department of Labor. Furthermore, Dr. Zaldivar's hourly charge is $400.00 (EX 6, pp. 3-8). In his substantive testimony, Dr. Zaldivar conceded that his x-ray finding of saroidosis, which he had made during the miner's lifetime, "was not a very good diagnosis" (EX 6, p. 11). Based upon his evaluation of the available medical data, including postmortem evidence, Dr. Zaldivar reiterated the opinion which he had set forth in the report, dated July 28, 2004, as outlined above. Moreover, Dr. Zaldivar rejected Dr. Jelic's opinion, "because an acute exacerbation of chronic bronchitis doesn't kill anyone." (EX 6, p. 26). Furthermore, Dr. Zaldivar credited the pathology finding of relatively mild micronodular pneumoconiosis, as reported by Dr. Oesterling (EX 6, 32).[9] Following his further discussion of the evidence, Dr. Zaldivar agreed that the miner had simple pneumoconiosis and was a "respiratory cripple" at the time of his death. However, Dr. Zaldivar opined that the miner's death was wholly unrelated to coal worker's pneumoconiosis (EX 6, pp. 31-38).

In a supplemental report, dated October 13, 2004, Dr. Zaldivar reviewed additional information supplied by Employer's representative, and, in particular, analyzed Dr. Rasmussen's report, dated October 7, 2004 (EX 7). In particular, Dr. Zaldivar provided a detailed analysis of Dr. Rasmussen's opinion, and challenged the medical literature cited therein, as not supporting

[9] Although not cited by Dr. Zaldivar, a similar pathology finding was reported by Dr. Jarboe (EX 4).

Dr. Rasmussen's underlying conclusions. On the other hand, Dr. Zaldivar cited other medical literature to support his own conclusions.
Following his extensive analysis, Dr. Zaldivar stated:

> In this specific case of Mr. Sheppard when faced with a very significant smoking habit as given by himself during his examinations, one of which was conducted by me, emphysema was found at autopsy. And this is not unexpected in an individual who had smoked as much as he did. Cancer of the lung was also found and certainly no one has ever implied any relationship between cancer of the lung and mining. Cancer of the lung was also the result of his smoking habit. The pneumoconiosis found was described by Dr. Oesterling as being minimal. Dr. Jelich (sic) did not describe the pneumoconiosis in any manner, whether mild or severe. The disagreement between Dr. Jelich (sic) and Dr. Oesterling is about whether a single lesion of complicated pneumoconiosis was present or not and this I leave up to the pathologist to decide. As I stated in my deposition, a one, two, three or even four centimeter lesion, in itself, is not sufficient to produce any pulmonary dysfunction at all given the size and reserve present in the lungs. The importance is the amount of pneumoconiosis present. The greater the amount of dust within the lungs the greater the chances of pulmonary impairment to be present causing serious breathing difficulties. As described by Dr. Oesterling, the amount of dust present in the lungs, in this case, was very small and itself insufficient to produce the emphysema, which was ascribed to smoking.
>
> In conclusion, after carefully reviewing the report of Dr. Rasmussen, my opinion remains exactly the same as I gave in the deposition and as I had previously given in this case. This opinion is based on what has up to now been described in the medical literature.

(EX 7, pp. 6-7).

Discussion and Applicable Law

As summarized in my Decision and Order Denying Benefits, dated November 20, 2001, and affirmed by the Board, the preponderance of the x-ray evidence which was submitted in conjunction with the miner's claims was negative for pneumoconiosis. Furthermore, I also found that the medical opinion evidence which had previously been submitted did not establish the presence of pneumoconiosis. The Board declined to address that issue, and, instead, affirmed the denial of benefits based upon Claimant's failure to establish disability causation (DX 5).

Notwithstanding the foregoing, the pathology evidence and recent medical opinion evidence submitted in this survivor's claim clearly establishes the presence of pneumoconiosis under §718.202(a). Furthermore, the Employer has failed to rebut the presumption that the disease arose from Mr. Sheppard's more than ten years of coal mine employment. 20 C.F.R. §718.203 and §718.302. However, in order to be eligible for benefits, Claimant must also establish that the miner's death was due to pneumoconiosis, as provided in the Act and applicable regulations.

Death due to Pneumoconiosis

Since the claim was filed after January 1, 1982, the issue of death due to pneumoconiosis is governed by §718.205(c), as amended, which states, in pertinent part:

> For the purpose of adjudicating survivor's claims filed on or after January 1, 1982, death will be considered to be due to pneumoconiosis if any of the following criteria is met:
>
> (1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or
>
> (2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
>
> (3) Where the presumption set forth at §718.304 is applicable.
>
> (4) However, survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death.
>
> (5) Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death.

20 C.F.R. §718.205(c).

As outlined above, there is no medical evidence which establishes that pneumoconiosis was the immediate and/or primary cause of the miner's death. Therefore, I find that Claimant has failed to establish "death due to pneumoconiosis" under §718.205(c)(1). However, I must weigh the conflicting evidence regarding the presence of *complicated* pneumoconiosis. If established, such a finding would invoke the irrebuttable presumption of death due to pneumoconiosis under §718.304. *See* 20 C.F.R. §718.205(c)(3). Furthermore, if Claimant failed to establish *complicated* pneumoconiosis, I must also consider whether the miner's *simple* coal worker's pneumoconiosis substantially contributed and/or hastened the miner's death. *See* 20 C.F.R. §718.205(c)(2), (4), (5).

For the purpose of rendering this decision, the more relevant evidence consists of the miner's death certificate (DX 15) and the recent medical opinions of Drs. Jelic (DX 17), Figueroa (CX 2), Oesterling (EX 1, 8; CX 4), Bush (EX 2), Jarboe (EX 4), Rasmussen (CX 1), and Zaldivar (EX 3, 6, 7), respectively.

As stated above, the death certificate was signed by Dr. Figueroa, who performed surgery on Mr. Sheppard less than one month prior to the miner's death, and who apparently was the surgeon for the miner's autopsy. However, it is unclear whether the autopsy findings were

available to Dr. Figueroa prior to the completion of the death certificate. Furthermore, the underlying bases for the findings contained in the death certificate are not provided. Moreover, I find that Dr. Figueroa's cursory report is also poorly reasoned and documented. Therefore, I accord the death certificate and Dr. Figueroa's opinion little weight (DX 15; CX 2).[10]

Of the remaining physicians, Dr. Jelic is the only one who diagnosed *complicated* pneumoconiosis. Furthermore, Drs. Jelic and Rasmussen are the only physicians who opined that the miner's pneumoconiosis and/or coal mine dust exposure substantially contributed to and/or hastened the miner's death. On the other hand, Drs. Oesterling, Bush, Jarboe, and Zaldivar found that Mr. Sheppard has simple pneumoconiosis; and, that neither pneumoconiosis nor Mr. Sheppard's coal mine dust exposure caused, contributed, and/or hastened the miner's death.

Upon consideration of the relevant evidence, I first note that Dr. Jelic's credentials are not included in the record. On the other hand, Drs. Oesterling and Bush are both well-credentialed, Board-certified pathologists. This weighs in favor of the latter pathologists. Dr. Rasmussen is Board-certified in various fields and also has extensive experience in the field of pulmonology. Drs. Jarboe and Zaldivar are both Board-certified pulmonary specialists. Despite Dr. Rasmussen's lack of Board-certification in pulmonary medicine, I find that his qualifications are roughly comparable to those of Drs. Jarboe and Zaldivar. Therefore, the respective qualifications of these three physicians are not dispositive.

In the "Order on Reconsideration Granting Claimant's Request for Post-Hearing Submission of Evidence," dated December 23, 2004, I allowed the belated submission of Dr. Oesterling's deposition by Claimant, and the submission of a rehabilitative report by Employer. In so ruling, I expressed concern that certain aspects of Dr. Oesterling's deposition appeared to undermine his initial report, and that part of his opinion may be contrary to the Act.

In *Daugherty v. Dean Jones Coal Company,* 895 F.2d 130 (4th Cir. 1989), the Fourth Circuit held that a pathology report prepared by a physician who examined biopsy specimen should not have been completely invalidated simply because it was based on lymphoid tissue, and not lung tissue. In so finding, the Court stated that lymphoid tissue from the hilar region "may be sufficient to sustain a determination of the presence or absence of anthracosis." Accordingly, the Court remanded the case for further consideration, and to allow for the introduction of additional evidence concerning the existence of anthracosis.

Having carefully considered Dr. Oesterling's deposition testimony, in conjunction with his initial and rehabilitative report, and the *Daugherty* case, I find that his opinion does not conflict with the Act and applicable case law. First, I note that the Court's ruling does not require that a physician diagnose pneumoconiosis based simply upon findings of anthracosis in the hilar lymph nodes. Moreover, unlike the *Daugherty* case, Dr. Oesterling reviewed slides of the hilar lymph nodes *and the lungs*. Furthermore, as stated above, Dr. Oesterling's conclusion that *complicated* pneumoconiosis was not present, is based not only on the location of the larger nodule, but also the fact the core of the nodule is hemosiderin. Furthermore, Dr. Oesterling

[10] I also note that the testimony by Claimant's daughter, Terena Surgoine, regarding statements allegedly made by Dr. Figueroa to her prior to the miner's death does not cure the underlying defects in his report.

clarified his opinion in a supplemental report, in which he clearly stated that the *size* of the nodule found in the hilar lymph node could be misconstrued as complicated coal worker's pneumoconiosis. Moreover, Dr. Oesterling reiterated that based upon its location, and the fact that the central core of the nodule is hemosiderin, it is *not* complicated pneumoconiosis.

Similarly, Dr. Bush also found that the pathology evidence only established a mild degree of simple coal worker's pneumoconiosis, based upon his analysis of the autopsy tissue and other evidence.

In addition to the superior credentials of Drs. Oesterling and Bush in the field of pathology, I also note that they provided more details, including specific measurements regarding the sizes of the nodules, in their microscopic descriptions. In contrast, Dr. Jelic did not include any measurements of nodules in his microscopic description. Furthermore, Dr. Jelic's provided little analysis for his conclusion that the miner's death was due to pneumoconiosis among various other conditions. In view of the foregoing, I accord greater weight to the opinions of Drs. Oesterling and Bush than that of Dr. Jelic, despite his status as the prosector. *See, Bill Branch Coal Corp. v. Sparks*, 213 F.3d 186 (4th Cir. 2000)(where the Court held that it was error to credit the prosector's report over those of reviewing physicians solely because he had access to the whole body).

In weighing the conflicting opinions of the remaining non-pathologists, I accord greater weight to the opinions of Drs. Zaldivar and Jarboe than that of Dr. Rasmussen. In so finding, I note that Dr. Rasmussen's recent report does not even address whether the miner suffered from complicated or simple pneumoconiosis. Moreover, Dr. Rasmussen acknowledges that Drs. Oesterling and Bush are highly competent pathologists who found that coal mine dust played no role in the miner's death. Furthermore, Dr. Rasmussen reports that Drs. Zaldivar and Jarboe also found no relationship between coal mine dust exposure and the miner's death. Nevertheless, citing medical literature, Dr. Rasmussen concludes that the miner's pneumoconiosis was a significant contributing factor in his disabling lung disease and death. I note, however, that Dr. Rasmussen's recent report does not discuss the miner's lifetime cigarette smoking history, and provides minimal discussion of the miner's other medical problems. Dr. Rasmussen's report is primarily a list of medical articles which purports to establish that it is scientifically impossible to exclude coal mine dust as a contributing factor in Mr. Sheppard's disabling condition and death. Accordingly, Dr. Rasmussen opined that it was a significant contributing factor in the miner's disabling lung disease and ultimately his death. However, Dr. Zaldivar, in his supplemental report, provided a far better documented and reasoned medical opinion, which discussed the medical literature, and undermines Dr. Rasmussen's conclusions. Furthermore, I find that Dr. Zaldivar's overall analysis of the evidence, as set forth in his recent reports and deposition testimony, is more detailed, and better reasoned and documented. Moreover, it is also buttressed by the well-reasoned report by Dr. Jarboe.

In summary, I accord no weight to the death certificate and/or Dr. Figueroa's poorly documented, cursory statement. Furthermore, I accord greater weight to the opinions of Drs. Oesterling, Bush, Zaldivar, and Jarboe than those of Drs. Jelic and Rasmussen. Accordingly, I

find that Claimant has not met her burden of establishing death due to pneumoconiosis under the provisions of 20 C.F.R. §718.205(c)(2), (3), (4), (5), or by any other means.[11]

Conclusion

The evidence shows that Mr. Sheppard had *simple* pneumoconiosis which arose from his 33 years of coal mine employment. However, it does not establish that pneumoconiosis caused, substantially contributed to, or hastened the miner's death. Therefore, I find that the Claimant is not entitled to benefits under the Act and applicable regulations.

Attorney's Fees

The award of an attorney's fee under the Act is permitted only in the cases in which Claimant is found to be entitled to benefits. Since benefits are not awarded in this case, the Act prohibits the charging of any fee to the claimant for services rendered to him in pursuit of this claim.

ORDER

It is ordered that the claim of Shelby J. Sheppard, surviving spouse of John E. Sheppard, Jr., for black lung benefits under the Act is hereby **DENIED.**

A

RICHARD A. MORGAN
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. 725.481, any party dissatisfied with this Decision and Order may appeal to the Benefits Review Board within 30 days from the date of this Decision and Order, by filing a notice of appeal with the **Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601.** A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room B2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.

[11] Finally, I note that the medical evidence presented in conjunction with the miner's claims does not support Claimant's position. The x-ray evidence was conflicting, and the preponderance of such evidence was negative for pneumoconiosis. Moreover, even the *positive* x-ray interpretations, if credited, only established *simple* pneumoconiosis. Furthermore, the previously submitted medical opinion evidence did not address the "death due to pneumoconiosis" issue.